serves as standard preparation of CSF in our laboratory[7]. HLCM was diluted at 1:16 to avoid excessive stimulation which might mask inhibition by somatostatin. Somatostatin at 0.05/0.5/5 µg/ml did not influence colony growth; 50 µg/ml however reduced colony formation (table 4). Since lymphocyte culture supernatants are diluted at 1:10 by transfer into the bone marrow culture medium, the remaining activity of somatostatin is far too low to cause the reduced colony growth observed with somatostatin-containing lymphocyte culture supernatants.

Since these experiments exclude direct suppression of somatostatin on CFU_C, reduced production or impaired release of CSF from PHA-treated lymphocytes must be responsible for our observations. Thus, somatostatin interferes with CSF elaboration, but does not affect DNA synthesis. The possibility however exists that assays for CSF release and DNA synthesis relate to different lymphocyte subpopulations. In this case, somatostatin would preferentially inhibit the CSF-releasing subpopulation.

It has been demonstrated that CSF production from mitogen-treated lymphocytes requires active protein synthesis[8]. Somatostatin is believed to inhibit ribosomal protein synthesis by competing with C-AMP, which activates protein-kinases by phosphorylation[9]. Accordingly, the inhibitory effect of somatostatin on rat liver protein synthesis becomes more pronounced by elevating the extracellular concentration of C-AMP in the cultures[10]. The demonstration of this effect in lymphocyte cultures, however, is impossible, since the addition of C-AMP to the culture medium markedly abolishes the PHA activation (Hirschorn[11] and own observations), probably due to loss of binding of PHA on the lymphocyte surface.

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Electrical activity of the pectoral muscles during gliding and flapping flight in the herring gull (*Larus argentatus*)[1]

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Summary. Electromyographic recording from the pectoral muscles of the herring gull during flight showed that very little muscle activity is associated with gliding flight. However, the integrated gliding potentials could be increased very considerably by loading the bird. The muscle activity during gliding and flapping flight are in accordance with the known energy requirements for these 2 types of flight.

It is well known that certain birds can glide for long periods of time. Birds such as gulls and storks can remain airborne for hours, or in the case of the albatross presumably for months, without landing or without having to resort to flapping flight. Some birds, such as African vultures, travel long distances by alternately soaring on up-drafts and gliding between up-drafts[3]. Gliding flight has been assumed to require considerably less energy than flapping flight. However, the only quantitative measurements of the energy requirements for gliding flight are those of Baudinette and Schmidt-Nielsen[4]. These workers found that the rate of oxygen consumption by the herring gull during gliding was about 2 times its resting level, whereas in level flapping flight of the laughing gull Tucker[5] found it to be 6–8 times the resting level. This present study was undertaken to see how much muscular activity is necessary for gliding flight, and to relate this to the known metabolic requirements of the bird.

Materials and method. The electrical activity of the pectoralis major muscle was measured during gliding and flapping flight of the herring gull (*Larus argentatus*). Measurements were carried out mainly on 1 gull (herring gull No. 72, 748 g) which had been trained for flying in a wind tunnel. Such training is very time-consuming and consists of h-long daily sessions for a period of several weeks. In order to obtain gliding flight the wind tunnel was tilted at an angle of 7° below the horizontal with a wind speed of 11.5 m sec^{-1}. Measurements were carried out on other birds, (laughing gull, *Larus atricilla,* and North American black vulture, *Coragyps atratus*) which also had been trained to fly in the wind tunnel. The results were essentially the same as for the herring gull.

EMG recordings were made from 3 insulated copper electrodes inserted into the muscle in a triangular configuration. Each copper wire (0.1 mm diameter) was first pushed through a syringe needle, and after removing the end 2 mm of the insulation, the tip of the wire was bent over to form a barb. The needle was inserted into the muscle and immediately withdrawn, leaving the wire electrode in the desired position. The distance between the 3 wires in the muscle was approximately 6 mm. These were connected to a small preamplifier (weight 23 g) which was mounted on the back of the bird with 1 of the wires connected to ground. The signal was further amplified to give an overall amplification of 1.3×10^5. Following this it was processed to give a rectified compressed signal plus a rectified linear signal. The former was obtained by putting it through a square rooter. The latter was obtained by putting it through the square rooter followed by a squarer, followed by a limiter (figure 1). This additional circuitry was necessary because a gain that was suitable for recording gliding potentials was not suitable for recording flapping potentials. In particular, the limiter was necessary to prevent overdriving of the recording system should the bird suddenly change from gliding to flapping flight.

The processed signals, the linear and the compressed, were recorded on separate channels of an fm tape recorder. A 3rd channel was used to mark the periods of stable gliding. The signals were recorded at a tape speed of 38 cm sec^{-1}, and eventually played back and recorded on paper using a

Mean integrated values in arbitrary units (dimensions mV sec^{-1}) of recorded potentials from the pectoral muscle of herring gulls during rest and during gliding flight

Recording	Resting	Gliding	
		Amplifier only (23 g)	Amplifier + weight (123 g)
1	34 ± 9 (7)	100 ± 34 (16)	241 ± 38 (20)
2	36 ± 3 (15)	83 ± 25 (31)	185 ± 43 (19)
3	22 ± 2 (14)	72 ± 18 (25)	206 ± 51 (30)
Mean	30	89	211

Values given ± the SD (Student's t-test). The number of times the traces were sampled are given in parentheses.

Brush model 220 recorder at a tape speed of 2.4 cm sec^{-1}. The 1st channel was used to display either the compressed signal or the linear signal continuously integrated with an RC time constant of 1 sec. To obtain mean numerical values for potentials during gliding, the integrated signal was sampled with a data acquisition system at intervals of 62.5 msec real time. The measurements were made under different conditions, as follows: resting, that is to say with the bird standing in the wind tunnel, gliding with only preamplifier, gliding with the preamplifier plus a 100-g load, flapping flight with the preamplifier, and flapping flight with preamplifier plus the 100-g load. Resting recordings were taken before, after, and between flights. Gliding potentials were analysed only for periods of stable gliding.

Results and discussion. Recording made during rest and during flight are shown in figure 2. The resting levels are quite low, and heart beats are clearly visible at regular intervals. The recorded resting signal could be due to a small amount of motor unit recruitment necessary to maintain 'muscle tone', or to extraneous electronic noise in the recording system. During gliding flight, and especially in flapping flight, the signal is greatly increased.

Mean value for the integrated signals during resting and gliding are listed in the table. During gliding flight (unloaded bird carrying only the amplifier) the potentials in the pectoral muscles increased approximately 3fold, relative to the resting potentials. Increasing the load by an additional 100 g (13% of the body weight) gave a noticeable increase in muscle activity, with the mean integrated potentials more than doubled, relative to the bird with amplifier only.

Compared to the gliding potentials, even those in the loaded bird, the flapping potentials were very large. Each downbeat of the wing was associated with a train of spikes, as shown in figure 2,D. It will be seen that such a train consisted of about 50 spikes, which are all much higher than the spikes seen during gliding.

These observations show that gliding in the herring gull does involve some muscular activity in the pectoral muscle, and that additional loads increase this activity during flapping flight, however, the gliding potentials are quite low.

The results are in agreement with observed metabolic requirements for gliding and for flapping flight, as men-

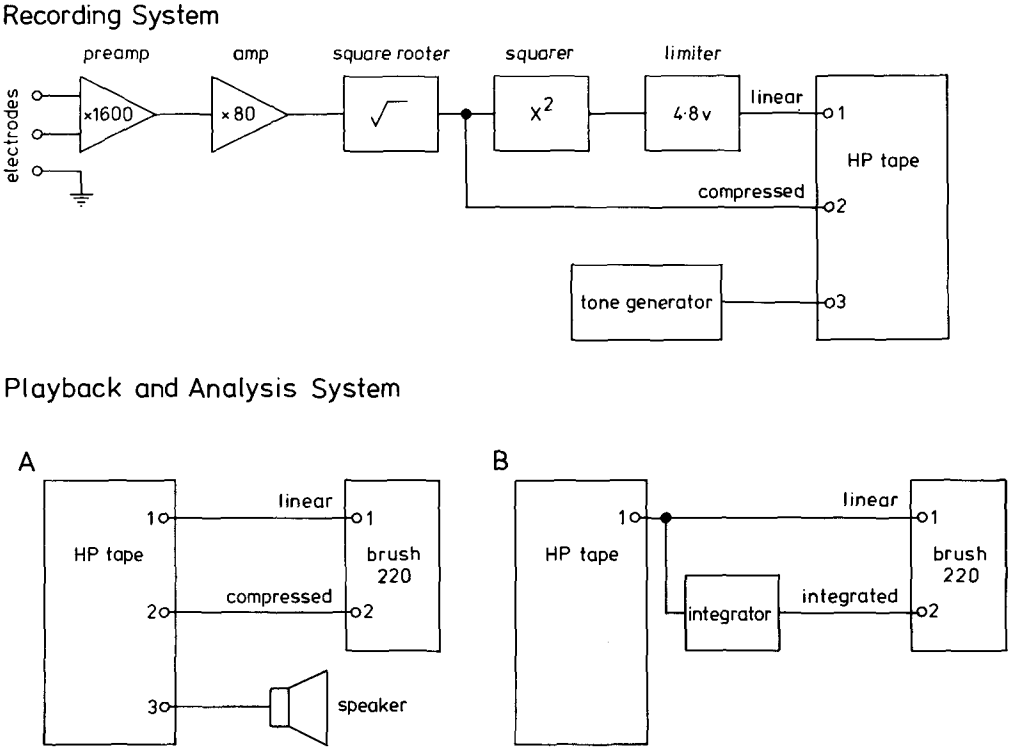

Fig. 1. Diagram of the systems used for recording and analyzing electrical potentials from birds during gliding and flapping flight. The preamplifier weighing 23 g is carried on the back of the bird. This was connected to the amplifier and the rest of the circuitry using light flexible shielded cable.

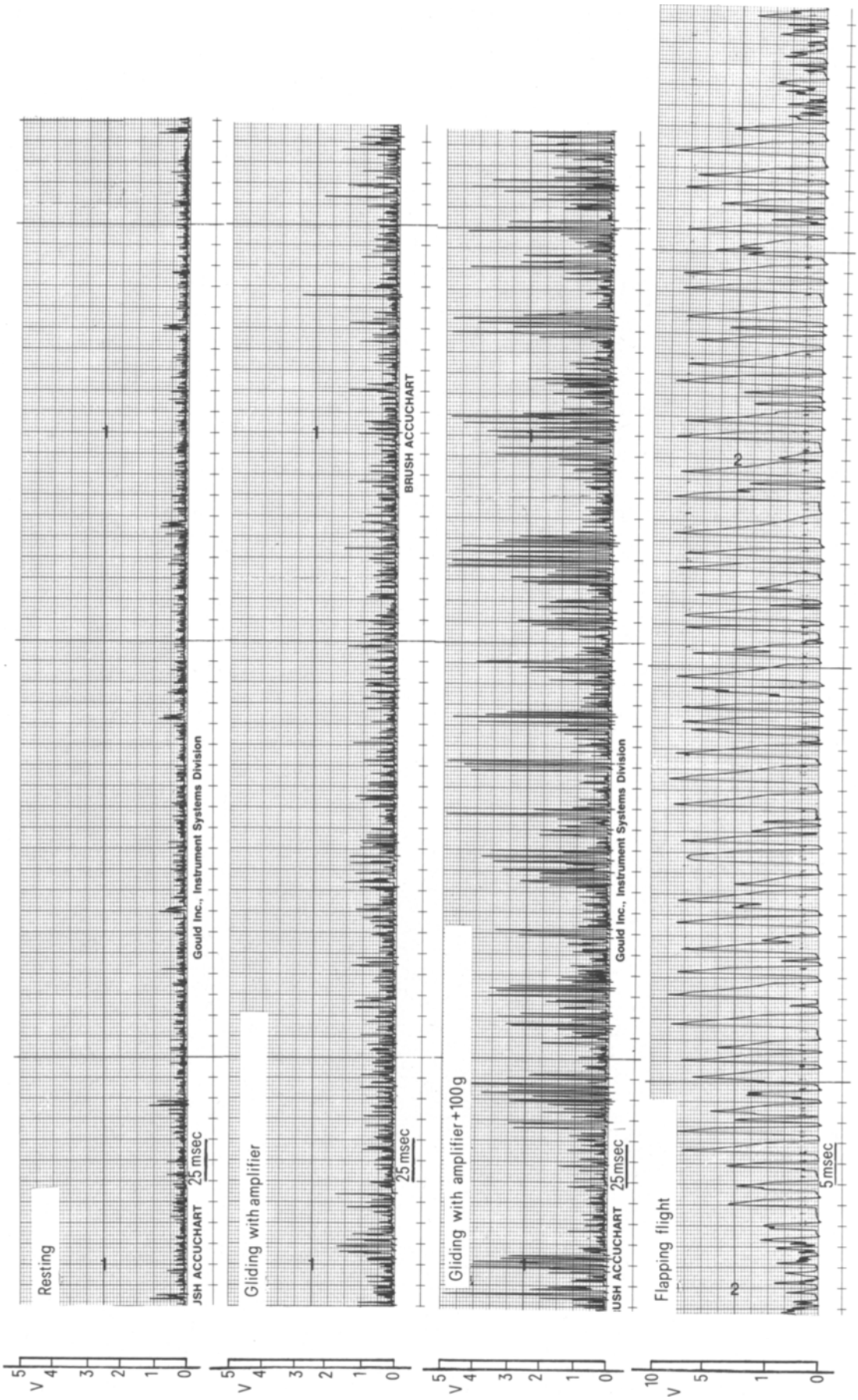

Fig. 2. Samples of traces showing the electrical potentials from the pectoralis muscle of the herring gull during resting, gliding and flapping flight. In the case of gliding flight measurements were made from the herring gull carrying the amplifier only (23 g) and carrying the amplifier plus a 100-g weight. The trace for flapping flight which shows the period just before downstroke to just after a downstroke was recorded using non linear amplification as described in the text. A faster time base was used in order to visualize the individual potential. The calibrations shown are for the measured potentials (not actual potentials) and are in V.

tioned in the introduction above. The recorded mean integrated potential for flapping flight was approximately 500 mV, against about 90 mV for gliding flight of the unloaded bird. Bearing in mind that gliding flight primarily involves isometric contraction (the energy turnover in isometric contraction is less than in isotonic contraction[6]) and that during flapping flight the pectoral muscles are only activated during the down beat, the magnitude of the potentials are in accord with the measured oxygen consumption for these 2 types of flight.

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Intravital measurement of arteriolar pressure and tangential wall stress in normotensive and spontaneously hypertensive rats (established hypertension)

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Summary. Under intravital conditions, intravascular pressures of mesenteric resistance vessels were measured in normotensive (NR, mean blood pressure 92 mm Hg) and spontaneously hypertensive rats (SHR, 161 mm Hg) being elevated over all by about 75%; the tangential wall stress ($\sigma = p \cdot r/h$; p represents the intravascular pressure and r/h the ratio of internal radius to wall thickness) was found to be increased by 120–140% in SHR.

The tangential (circumferential) wall stress as a basic haemodynamic parameter[2] was recently determined in cat mesentery vascular bed by direct measurement of intravascular pressures[3,4]. For normotensive control rats (NR) and spontaneously hypertensive animals (SHR), intravital pressure measurements have been performed in the microvasculature of the cremasteric muscle[5]. These measurements could technically be done only in young animals, and therefore the SHR studied were prehypertensive and their blood pressure was only slightly elevated (116 mm Hg). By using the mesentery microvascular bed, measurements in older SHR suffering from established hypertension (161 mm Hg) could be performed[6]. The studies presented attempt to answer the question whether intravascular pressures in the smallest precapillary arterioles would be reduced to a normal level, perhaps for the sake of protecting the capillary bed. It was of further interest to study the effect of pressure elevation, 'rarification of resistance vessels'[6] and wall hypertrophy[7] on the tangential wall stress in precapillary arterioles of spontaneously hypertensive rats.

Materials and methods. The experiments were performed in 16–20-week-old sex-matched Kyoto-Wistar rats of the Okamoto-Aoki strain. The SHR had an average b.wt of 209±10 g and a systemic blood pressure measured in the carotid artery of 161±4 mm Hg, the NR reached 225±7 g and 92±9 mm Hg, respectively. The animals were anaesthetized with chloralose-urethane (10 or 50 mg/ml 0.9% NaCl/100 g b.wt). The mesentery preparation was suffused with modified Tyrode's solution containing 1% dextran[8]. Using dextran in the suffusing solution, we could not see any anaphylactoid response in the rat microcirculation like vasodilatation, elevation of red cell velocity or formation of oedema, which is reported for intravascular application of dextran[9].

The preparation was allowed to stabilize for about 30 min after surgery. The tissue was transilluminated by a 150 W Halogen light source and simultaneously displayed on a video monitor and on video tape. Inside vessel diameters were measured on line by the video-angiometer[10] or by using a ruler (optico-electronic magnification × 1100). Using intravital microscopic techniques, the in vivo determination of the real outer diameters of microvessels is actually difficult or impossible. Therefore, the muscular media layer of the arterial wall[11] was taken, representing the most relevant structure of the vessel wall, especially of the hypertrophic wall in hypertensive animals[7].

Pressures in microvessels were measured by using a modified servo-null system after Wiederhielm. We used micropipets with an outer tip diameter of 0.3–3.0 µm. Besides microscopic observation, the criterion for a successful measurement of intravascular pressures was a corresponding systolic-diastolic amplitude of microvascular and systemic pressures. Results obtained under an unsteady level of blood pressure were discarded. The arterioles to be measured were classified according to their branching order: the precapillary arterioles (A 4) were fed by terminal arterioles (A 3), which arose from large arterioles (A 2).

Results and discussion. Figure 1 shows averaged data of diameters and pressures in microvessels. The mean values of systemic pressure for NR was 92 mm Hg and for SHR 161 mm Hg representing a difference of 75%. The mean arteriolar pressure (i.e. in A 4, A 3 and A 2 together) ranged from 34 mm Hg in NR to 55 mm Hg in SHR, being a difference of 61%. This confirms the findings of pressure distribution in the cat mesentery microvascular bed, where a correlation between systemic and microvascular pressures in arterioles below 60 µm inner diameter could not be found[12]. Compared with the data obtained in prehypertensive SHR[5], our measurements, performed in SHR during the established phase of hypertension, showed pressures even twice as high. The mean blood pressure of the NR, however, differed only by 3% under the same anaesthesia. Consequently, hypertension in its established phase led to a distinct elevation of the precapillary pressures. Referring to this, the observation of a decreased number of precapillary arterioles in SHR should be taken into account, which is shown for prehypertensive SHR[13] as well as for SHR with established hypertension[14]. Measuring tangential wall stress in these particular arterioles further confirms this aspect.